https://archive.fo/6uBY3

The health consequences of race mixing

Posted by J Richards on Monday, 06 June 2005 22:38.

The health consequences of race mixing among living organisms could be neutral, beneficial or harmful. This issue has been difficult to address in humans, but data from random, population-based studies have started coming in, and here we consider the first such dataset.
The data in question come from J. Richard Udry's National Longitudinal Study of Adolescent Health, which sampled a random, nationally representative school-based sample of U.S. adolescents in grades 7 through 12, during 1994-1995. [1]

Paper-based questionnaires were completed by 83,135 adolescents; a random sub-sample of these individuals plus some individuals in the school roster that had not completed the paper-based questionnaire, totaling 18,924 adolescents, were interviewed at home. The data are reported for the home-interviewed sample.

Racial classifications are based on self-report; the participants were allowed to pick multiple racial categories to describe themselves. Table 1 shows participant characteristics. The participants were asked if they were Hispanic/Latino, but this was not considered to be a racial category. 86% of those who only chose "other" race also described themselves as Hispanic, and 46% of those who described themselves as Hispanic only chose "other" race. 72% of those identifying as American Indian also picked another racial category, usually white.

TABLE 1—Race Combinations (Includes Missing Race)

Race/ethnicity	School Questionnaire (Percentage of Total)			Home Interview (Percentage of Total)		
	Frequency	Unweighted	Weighted	Frequency	Unweighted	Weighted
White only	46 364	55.8	58.2	11 032	58.3	69.8
Black only	13 530	16.3	17.9	3 867	20.4	15.2
Asian only	4 133	5.0	3.5	1 289	6.8	3.4
American Indian only	1 275	1.5	1.7	211	1.1	0.9
Other only	5 817	7.0	5.6	1 553	8.2	6.4
White and Black	416	0.5	0.5	138	0.7	0.5
White and Asian	583	0.7	0.6	130	0.7	0.5
White and American Indian	1 573	1.9	2.0	268	1.4	1.6
White and Other	1 024	1.2	1.2	109	0.6	0.5
Black and Asian	294	0.4	0.4	26	0.1	0.1
Black and American Indian	590	0.7	0.8	91	0.5	0.3
Black and Other	151	0.2	0.2	50	0.3	0.1
Asian and American Indian	83	0.1	0.1	6	0.0	0.0
Asian and Other	294	0.4	0.2	19	0.1	0.1
American Indian and Other	93	0.1	0.1	18	0.1	0.1
≥3 categories	1 045	1.3	1.3	81	0.4	0.3
Missing race	5 870	7.1	5.8	36	0.2	0.2
Total	83 135	100.0	100.0	18 924	100.0	100.0

The data are presented in terms of odds ratios and statistical significance, both of which are briefly explained in the next paragraph, which knowledgeable individuals can skip.

The odds ratio (OR) is the number of times something is more prevalent in one group compared to another. Thus, OR = 2.0 means a two-fold greater prevalence, OR = 1.3 means a 1.3-fold greater prevalence, OR = 1.0 means no difference, OR = 0.5 means half as prevalent, and so on. A statistically significant difference is a difference that most likely represents a genuine difference between the groups compared rather than a difference due to chance factors alone. If the probability that the difference obtained is due to chance is less than 5%, then the difference is typically considered statistically significant, which is denoted as $p < 0.05$; similarly, if the probability that the difference obtained is due to chance is less than 1%, then this is designated as $p < 0.01$. All statistically significant differences in Tables 2-4 ($p < 0.05$) are marked by an asterisk. On the other hand, due to inadequate sample size, genuine differences may not reach statistical significance in a study, although trends toward differences may be clear.

Table 2 shows that mixed-race individuals had an across-the-board higher incidence of health and behavior problems; the specific health problems addressed were assessed in terms of whether they occurred frequently or every day of the previous month.

TABLE 2—Health and Behavior: Odds Ratios for Mixed-Race Adolescents Compared With Single-Race Adolescents

Attribute	White	Black	American Indian	Asian	Other
General health					
Health fair/poor	1.69*	1.60*	0.84	1.68*	1.31
Wake up tired	1.12*	1.37*	1.26*	1.55*	1.74*
Skin problems	1.23*	1.63*	1.32*	1.18	1.38*
Headaches	1.19*	1.28*	1.12	2.11*	1.48*
Aches/pains	1.34*	1.48*	1.27*	1.98*	1.56*
Sleep problems	1.58*	1.63*	1.24	1.72*	1.33*
Depressed/blue	1.50*	1.94*	1.45*	1.46*	1.45*
Substance use					
Smoke	1.24*	2.17*	1.03	2.25*	1.39*
Drink	1.22*	1.66*	1.00	2.37*	1.17
Get drunk	1.10	2.09*	0.95	2.96*	1.33*
Access to guns[a]					
Gun easily available	0.84	1.12	1.75	1.57	1.74
Suicidality[a]					
Seriously considered	1.34*	1.50	1.10	1.84*	1.31
Sexual behavior[a]					
Ever had sex	1.50*	0.83	1.50	2.87*	1.16
School behavior[a]					
Skipped school ≥ 10 times	1.43	1.97	0.77	1.84	1.17
Repeated a grade[a]	1.64*	0.91	1.02	2.11*	0.89
Ever suspended[a]	1.94*	1.06	1.08	2.58*	0.82

[a]Home interview. Odds ratio is defined as (1 / [1 – rate for mixed race]) / (1 / [1 – rate for one race]).
*$P < .05$

Given the overall picture in Table 2, sample size limitations, and the fact that the sample is random and representative, all values in Table 2 that are clearly greater than 1.00 but not statistically significant can be considered as genuinely reflecting increased likelihood of health/behavior problems among mixed-race adolescents, and this also applies to Table 3.

Now, it may be that adverse outcomes vary by race combinations. To examine this issue, Table 3 compares various mixed-race combos with their single-race counterparts on health/behavior issues. The general pattern seen in Table 2 is also seen in Table 3, i.e., irrespective of which races are combined, there is an overall increase in health/behavior problems among mixed-race adolescents.

TABLE 3—Odds Ratios for Specific 2-Race Adolescents Compared With Single-Race Adolescents

Virgule	Health Fair/Poor	Wake Up Tired	Skin Problems	Headaches	Aches/ Pains	Sleep Problems	Depressed/ Blue	Regular Smoker	Regular Drinker	Drunk Regularly
White/Black										
White	1.36	0.86	1.22	0.93	0.93	1.09	1.53*	0.90	1.11	1.28
Black	1.05	1.23	1.65*	0.88	1.04	1.23	1.76*	2.66*	1.52*	2.09*
White/American Indian										
White	1.73*	1.10	1.24*	1.15	1.56*	1.77*	1.54*	1.54*	1.37*	1.14
American Indian	0.79	1.26*	1.35*	1.01	1.26*	1.33	1.40*	1.36*	1.13	1.04
White/Asian										
White	1.50*	1.28*	1.26*	1.16	0.94	1.58*	1.21	1.17	1.14	1.16
Asian	1.12	1.80*	1.08	1.78*	1.33*	1.54*	1.11	2.24	2.06*	1.97*
Black/American Indian										
Black	1.31	1.42*	1.24*	1.31	1.35*	1.61*	1.65*	1.03	1.12	1.02
American Indian	0.78	1.13	1.00	1.21	0.99	1.07	1.30	0.31*	0.68*	0.57*
Black/Asian										
Black	2.25*	1.38*	1.76*	1.23	1.25	2.11*	2.14*	3.44*	2.22*	4.08*
Asian	2.17*	1.36*	1.11	2.00*	1.60*	1.83*	1.70*	2.22*	2.92	4.25*
Asian/American Indian										
Asian	1.93	1.45	0.83	3.00*	4.64*	2.18*	1.26	1.83	2.00*	2.23
American Indian	1.19	1.18	1.05	1.71	2.65*	1.68	1.24	0.85	0.91	1.20

Note. Odds ratio is defined as (1 / [1 – rate for mixed race]) / (1 / [1 – rate for one race]).
*Different from mixed race at $P < .05$.

Let us address one confound in interpreting the data in Tables 2 and 3. It is commonly observed that a disproportionate number of white women who end up with black men are obese. Obese women tend to have elevated testosterone levels. [2, 3] Some portion of testosterone is converted to dihydrotestosterone (DHT), which amplifies the effect of testosterone at certain targets. [4] DHT is also one of the culprits in the genesis of acne. [5] Therefore, a higher incidence of, say, acne in black/white-mixed offspring with a white mother could simply reflect inheritance of the genetics of elevated androgens from the obese white mother rather than an effect of race mixing.

The point above needs to be considered in a more general scenario. When the races occupying the same geographic region differ in status, as in present-day Western societies, members of the high-status race who marry those of a lower-status race tend to have low status within their group. There are, of course, many examples to the contrary: Carmen Electra (mostly white) with Dennis Rodman (black), Heidi Klum (white) with Seal (black), and David Bowie (white) with Iman (Somali), among others. However, the aforementioned trend is clear, which raises the possibility that the negative correlates of race mixing are largely related to the unhealthiness of one or both parents rather than the deleterious effects of race mixing. This potential confound can be addressed by statistically controlling for demographic variables.

The authors controlled for age, sex, verbal IQ, grade point average, family structure (living with one or both parents), and family education; education can act as a proxy for socioeconomic status, and given an inverse relation between socioeconomic status and obesity in white women, [6] education can also act as a proxy for obesity in the white mothers of the mixed-race adolescents. Controlling for all these factors left the general picture seen in Tables 2 and 3 unchanged; some statistically significant odds ratios lost statistical significance, but the ones greater than 1.00 can still be considered significant for the reasons addressed above, and there were very few instances of greater-than-1.00 odds ratios diminishing to less than 1.00. Therefore, one can conclude that the negative health/behavior consequences of race mixing are related to race mixing per se rather than the variables controlled for.

The authors mention that the most common explanation of their find, which has also been reported by others using non-random samples, is that stress associated with identity conflict or struggle with identity formation is the culprit, but they also note that there is no proof in this regard. [1] Indeed, it is difficult to believe that struggle with identity formation is responsible for an across-the-board increase in health/behavior problems in mixed-race adolescents. How does struggle with identity formation lead to a higher incidence of skin problems?

An across-the-board increase in health problems should be evaluated in light of the basic architecture of physiological control, wherein the brain and the autonomic nervous system (ANS) are especially relevant. As is seen in the figure below, the ANS is involved in the autonomic (involuntary; automatic) regulation of organs, and its abnormalities could easily be behind Udry's data.

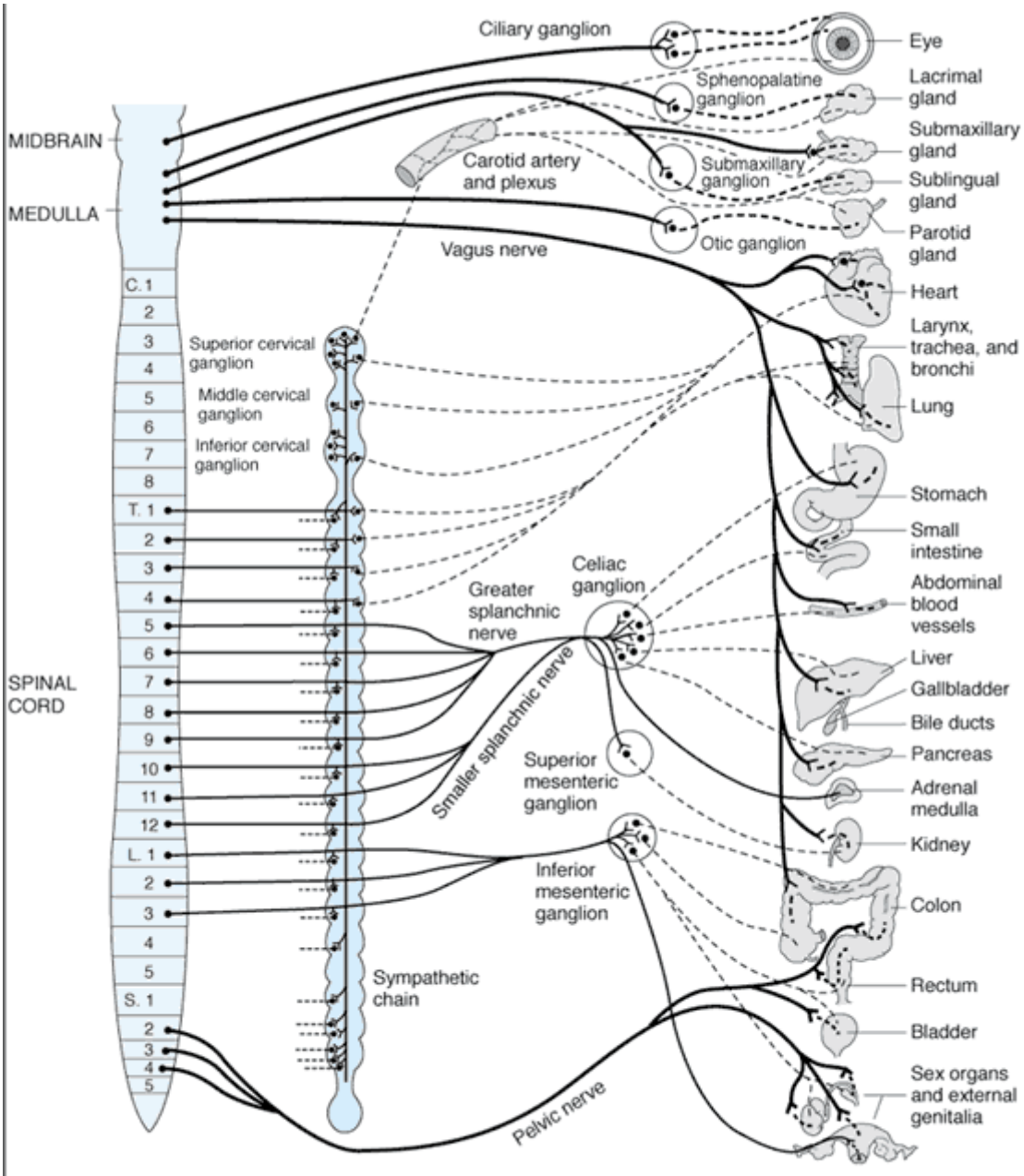

Figure 13-2. Diagram of the efferent autonomic pathways. Preganglionic neurons are shown as solid lines, and postganglionic neurons as dashed lines. The heavy lines are parasympathetic fibers; the light lines are sympathetic. (Modified and reproduced, with permission, from Youmans W: *Fundamentals of Human Physiology*, 2nd ed. Year Book, 1962.)

ANS abnormalities are implicated in behavior problems such as excessive aggression, [7-11] atopy (a group of diseases such as asthma, skin problems such as eczema and psoriasis, allergies, runny nose, sinusitis, migraine), [12-16] headaches, [17-22] and a variety of health problems (see any textbook of medical physiology). Further, damaging some portions of the brain in rats increases the likelihood of cocaine or stimulant dependence, [23, 24] and something similar undoubtedly applies to humans, too. Besides, a number of brain abnormalities are common to both depression and drug dependence, [25] and substance use is more prevalent among mentally ill individuals. [26]

Therefore, to the extent that race mixing may increase the likelihood of non-optimal genetic correlation structures, it may be expected to adversely affect organism-level physiological control, which may very well account for Udry's data.

Alternatively, since part of the ANS deals with stressors, chronic social stressors could themselves cause a number of health problems. In this regard, the data in Tables 2 and 3 do not vary as a function of whether the adolescent is living with one or two parents or as a function of family education, which can act as a proxy for socioeconomic status. Being teased/ostracized for being mixed race also requires consideration, but it cannot be argued that during the time period of the childhood years of the Americans examined, a substantial number of the mixed-race children would have experienced ostracism/discrimination due to being mixed race, especially since a number of them would have grown up in large metropolitan areas, which are known to be more diverse and tolerant. Moreover, the general preference in the African-American community is to prefer African-Americans with lighter skin, [27, 28] and lighter-skinned African-Americans also tend to have higher IQs than their darker counterparts (Table 4, [29]), which should reduce academic stress. In other words, black/white-mixed individuals, who would typically be assigned and raised as African-Americans, are not necessarily really worse off compared to their darker counterparts with respect to being ostracized/marginalized. Additionally, given white preference for Caucasian features and to the extent that Asian-Americans are envious of some of the facial features of whites, Asian/white-mixed individuals are less apt to be teased for their facial features than the less mixed Asians-Americans. And once again, there is no proof that struggle with identity formation explains Udry's data. Also, the typical mixed-race adolescent with a white parent should have no doubts as to whether he is white; he wouldn't look anything like a white person. Further, the data in Tables 2 and 3 do not vary as a function of age, which is relevant because the mixed-race individuals should have resolved their racial identity by their late teens.

On the other hand, there exist several examples in the animal literature where matings between more genetically distant individuals within the same species/different races result in offspring that are less healthy than the parents, on average, [30-37] and this cannot be blamed upon struggle with identity formation. There also exist examples of hybrid vigor, but nothing remotely close to hybrid vigor is seen in Udry's data.

Consider also the fact that the genetic correlation structures manifesting as different races are so distinct that when a computer was asked to classify DNA data (326 microsatellite markers) from 3,636 individuals self-identifying as either white, East Asian, African-American, or Hispanic, into clusters, without being told which DNA sample came from which racial group, the computer clustered the DNA data into 4 groups, classifying all but 5 individuals into the correct self-identified racial group. [38]

Therefore, a parsimonious explanation of the across-the-board negative health correlates of race mixing in Udry's data is that race mixing involving notably different races such as human races, by increasing the likelihood of non-optimal genetic correlation structures, increases the likelihood of deleterious effects.

Udry's data are compatible with the likelihood of race mixing improving one or more parental traits in some mixed-race offspring, who may be better off than both parents on multiple counts, provided that a greater number of mixed-race offspring are overall worse off than both parents. The former possibility is surely not implausible given that the tremendous racial and species diversity out there implies that nature does not rule out equally-well functioning/better functioning novel genetic correlation structures, which could be brought about by race mixing, though the chances of improvement would typically be slim if more distant races are involved.

Further, those identifying as mixed-race in this study would generally have greater genetic admixture than those identifying as single-race. It is seen in Table 2 that those identifying as mixed race have worse health than even populations known to be highly admixed (American black, Native American, Hispanic). This could be accounted for if one assumes that first-generation hybrids who have the worst health/behavior problems would disproportionately not be very successful in reproducing, i.e., the healthier mixed offspring could, within a few generations, set up a mixed-race population that is healthier, on average, compared to the first-generation hybrids, but for this mixed-race population to approach or exceed the overall health of the original single-race populations, it would take many generations of [naturally] weeding out the unhealthy and settling toward a novel population-typical genetic correlation structure that corresponds to good health (more on this and on hybrid vigor in a subsequent post).

To conclude, it is irresponsible for any scientific organization to pretend that race mixing has no adverse health effects and it is obviously inappropriate to portray race mixing as desirable or virtuous.

Extra stuff (for those interested in additional details):

Table 4 compares mixed-race adolescents with their single-race counterparts on several measures. The GPA (grade point average) and the PVT (picture vocabulary test) categories feature the percentage of individuals in the 75th percentile of the entire sample.

TABLE 4—Percentage of Adolescents With Individual and Family Attributes on School Questionnaire, by Race Ethnicity (Except PVT)

Virgule	GPA (High)	PVT (High)	College-Educated Parent	2-Parent Household
White/Black	24.19	18.20	39.46	52.77
White	32.27	26.98	41.30	78.41*
Black	15.45*	7.94*	35.39	47.25
White/American Indian	27.10	24.08	29.63	74.07
White	32.27*	26.98	41.30*	78.41*
American Indian	14.97*	10.29*	25.83	60.77*
White/Asian	37.58	23.08	52.36	73.54
White	32.27	26.98	41.30*	78.41
Asian	43.16	20.99	60.80	78.78
Black/American Indian	18.79	7.92	38.55	48.21
Black	15.45	7.94	35.39	47.25
American Indian	14.97	10.29	25.83*	60.77*
Black/Asian	18.57	[a]	35.81	47.76
Black	15.45	[a]	35.39	47.25
Asian	43.16*	[a]	60.80*	78.78*
Asian/American Indian	21.82	[a]	49.46	49.17
Asian	43.16*	[a]	60.80	78.78*
American Indian	14.97	[a]	25.83*	60.77

GPA = grade point average; PVT = Picture Vocabulary Test.
[a]Cell size too small.
*Different from mixed race at $P < .05$.

Some of the responses to self-identified race differed between the school-based and home-based assessments (16% of entire sample). Over 90% of the respondents self-identifying as white only, black only, or Asian only at school, self-identified as the same race at home; the changes involved adding/substituting "other" race. Somewhat less than half the respondents picking multiple races at school picked the same categories at the home interview, and to the extent that those identifying as multiracial in school identified as single race at home, given the picture in Table 2, these individuals are likely making the single-race individuals look worse. 34% of those identifying as American Indian only at school identified as white only at home, and 77% of those identifying as white/American Indian at school identified as white only at home, but American Indians were a miniscule percentage of the study sample. The inconsistent responses among the mixed-race adolescents may have resulted from context (school vs. home), assessment technique (paper-based vs. interview), ambiguity of question asking about race, or lack of a fully developed racial concept at the time of the assessment. [1]

Literature cited:

1. Udry JR, Li RM, Hendrickson-Smith J: Health and behavior risks of adolescents with mixed-race identity. Am J Public Health 2003, 93:1865-1870.
2. Garaulet M, Perex-Llamas F, Fuente T, Zamora S, Tebar FJ: Anthropometric, computed tomography and fat cell data in an obese population: relationship with insulin, leptin, tumor necrosis factor-alpha, sex hormone-binding globulin and sex hormones. Eur J Endocrinol 2000, 143:657-666.
3. Sowers M, Beebe J, McConnell D, Randolph J, Jannausch M: Testosterone concentrations in women aged 25-50 years: associations with lifestyle, body composition, and ovarian status. Am J Epidemiol 2001, 153:256-264.
4. Dadras SS, Cai X, Abasolo I, Wang Z: Inhibition of 5alpha-reductase in rat prostate reveals differential

regulation of androgen-response gene expression by testosterone and dihydrotestosterone. Gene Expr 2001, 9:183-194.
5. Thiboutot D: Acne: hormonal concepts and therapy. Clin Dermatol 2004, 22:419-428.
6. Stunkard AJ: Socioeconomic status and obesity. Ciba Found Symp 1996, 201:174-182; discussion 182-177, 188-193.
7. McBurnett K, Lahey BB, Rathouz PJ, Loeber R: Low salivary cortisol and persistent aggression in boys referred for disruptive behavior. Arch Gen Psychiatry 2000, 57:38-43.
8. van Goozen SH, Matthys W, Cohen-Kettenis PT, Thijssen JH, van Engeland H: Adrenal androgens and aggression in conduct disorder prepubertal boys and normal controls. Biol Psychiatry 1998, 43:156-158.
9. van Goozen SH, Matthys W, Cohen-Kettenis PT, Buitelaar JK, van Engeland H: Hypothalamic-pituitary-adrenal axis and autonomic nervous system activity in disruptive children and matched controls. J Am Acad Child Adolesc Psychiatry 2000, 39:1438-1445.
10. van Goozen SH, van den Ban E, Matthys W, Cohen-Kettenis PT, Thijssen JH, van Engeland H: Increased adrenal androgen functioning in children with oppositional defiant disorder: a comparison with psychiatric and normal controls. J Am Acad Child Adolesc Psychiatry 2000, 39:1446-1451.
11. van Goozen SH, Matthys W, Cohen-Kettenis PT, Gispen-de Wied C, Wiegant VM, van Engeland H: Salivary cortisol and cardiovascular activity during stress in oppositional-defiant disorder boys and normal controls. Biol Psychiatry 1998, 43:531-539.
12. Darsow U, Ring J: Neuroimmune interactions in the skin. Curr Opin Allergy Clin Immunol 2001, 1:435-439.
13. Harrison LC, Callaghan J, Venter JC, Fraser CM, Kaliner ML: Atopy, autonomic function and beta-adrenergic receptor autoantibodies. Ciba Found Symp 1982:248-262.
14. Moretti M, Varga G: [Psychosomatic dermatologic symptoms]. Orv Hetil 2000, 141:169-172.
15. Nabarro G, van Sandwijk FJ, van Vloten WA: [Constitutional eczema in the light of the biopsychosocial illness model]. Ned Tijdschr Geneeskd 1994, 138:231-234.
16. Nelson HS: What is atopy? Sidestepping semantics. Postgrad Med 1984, 76:118-120, 123-119.
17. Edvinsson L: Aspects on the pathophysiology of migraine and cluster headache. Pharmacol Toxicol 2001, 89:65-73.
18. Harle DE, Evans BJ: The optometric correlates of migraine. Ophthalmic Physiol Opt 2004, 24:369-383.
19. May A: [Headache attacks with ipsilateral autonomic symptoms]. Schmerz 2004, 18:370-377.
20. Ostertag D, Strittmatter M, Schimrigk K: [Autonomic dysfunction in migraine und tension-type headache—pilot study]. Schmerz 1998, 12:25-29.
21. Sanchez del Rio M, Reuter U: Pathophysiology of headache. Curr Neurol Neurosci Rep 2003, 3:109-114.
22. Shechter A, Stewart WF, Silberstein SD, Lipton RB: Migraine and autonomic nervous system function: a population-based, case-control study. Neurology 2002, 58:422-427.
23. Deminiere JM, Piazza PV, Le Moal M, Simon H: Experimental approach to individual vulnerability to psychostimulant addiction. Neurosci Biobehav Rev 1989, 13:141-147.
24. Schenk S, Horger BA, Peltier R, Shelton K: Supersensitivity to the reinforcing effects of cocaine following 6-hydroxydopamine lesions to the medial prefrontal cortex in rats. Brain Res 1991, 543:227-235.
25. Markou A, Kosten TR, Koob GF: Neurobiological similarities in depression and drug dependence: a self-medication hypothesis. Neuropsychopharmacology 1998, 18:135-174.
26. Regier DA, Farmer ME, Rae DS, Locke BZ, Keith SJ, Judd LL, Goodwin FK: Comorbidity of mental disorders with alcohol and other drug abuse. Results from the Epidemiologic Catchment Area (ECA) Study. Jama 1990, 264:2511-2518.
27. Bond S, Cash TF: Black beauty: Skin color and body images among African-American college women. J Appl Soc Psychol 1992, 22:874-888.
28. Hall RE: Bias among African-Americans regarding skin color: implications for social work practice. Res Soc Work Practice 1992, 2:479-486.
29. Rushton JP, Jensen AR: Thirty years of research on race differences in cognitive ability. Psychol Public Policy Law 2005, 11:235-294.
30. Aspi J: Inbreeding and outbreeding depression in male courtship song characters in Drosophila montana. Heredity 2000, 84 (Pt 3):273-282.
31. Edmands S, Feaman HV, Harrison JS, Timmerman CC: Genetic consequences of many generations of hybridization between divergent copepod populations. J Hered 2005, 96:114-123.

32. Garnier-Gere PH, Naciri-Graven Y, Bougrier S, Magoulas A, Heral M, Kotoulas G, Hawkins A, Gerard A: Influences of triploidy, parentage and genetic diversity on growth of the Pacific oyster Crassostrea gigas reared in contrasting natural environments. Mol Ecol 2002, 11:1499-1514.
33. Miller LM, Close T, Kapuscinski AR: Lower fitness of hatchery and hybrid rainbow trout compared to naturalized populations in Lake Superior tributaries. Mol Ecol 2004, 13:3379-3388.
34. Neff BD: Stabilizing selection on genomic divergence in a wild fish population. Proc Natl Acad Sci U S A 2004, 101:2381-2385.
35. Peer K, Taborsky M: Outbreeding depression, but no inbreeding depression in haplodiploid Ambrosia beetles with regular sibling mating. Evolution Int J Org Evolution 2005, 59:317-323.
36. Thornhill R, Moller AP: Developmental stability, disease and medicine. Biol Rev Camb Philos Soc 1997, 72:497-548.
37. Livshits G, Kobyliansky E: Lerner's concept of developmental homeostasis and the problem of heterozygosity level in natural populations. Heredity 1985, 55 (Pt 3):341-353.
38. Tang H, Quertermous T, Rodriguez B, Kardia SLR, Zhu X, Brown A, Pankow JS, Province MA, Hunt SC, Boerwinkle E, et al: Genetic structure, self-identified race/ethnicity, and confounding in case-control association studies. Am J Hum Genet 2005, 76:268-275.